PPARs and ERRs: molecular mediators of mitochondrial metabolism.

Journal: Curr Opin Cell Biol

Publication Year: 2015

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PubMed link: 25486445

Funding Grants: Metabolically-driven epigenetic changes in iPSC reprogramming

Public Summary:
Since the revitalization of “the Warburg effect”, there has been great interest in mitochondrial oxidative metabolism, not only from the cancer perspective but also from the general biomedical science field. As the center of oxidative metabolism, mitochondria and their metabolic activity are tightly controlled to meet cellular energy requirements under different physiological conditions. One such mechanism is through the inducible transcriptional co-regulators PGC1α and NCOR1, which respond to various internal or external stimuli to modulate mitochondrial function. However, the activity of such co-regulators depends on their interaction with transcriptional factors that directly bind to and control downstream target genes. The nuclear receptors PPARs and ERRs have been shown to be key transcriptional factors in regulating mitochondrial oxidative metabolism and executing the inducible effects of PGC1α and NCOR1. In this review, we summarize recent gain- and loss-of-function studies of PPARs and ERRs in metabolic tissues and discuss their unique roles in regulating different aspects of mitochondrial oxidative metabolism.

Scientific Abstract:
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